UNIVERSITÀ DEGLI STUDI FIRENZE

FLORE
Repository istituzionale dell'Università degli Studi di Firenze

Transient Hepatic Parenchymal Enhancement detected at dynamic imaging: A short instruction manual for the clinician.

Questa è la Versione finale referata (Post print/Accepted manuscript) della seguente pubblicazione:

Original Citation:

Transient Hepatic Parenchymal Enhancement detected at dynamic imaging: A short instruction manual for the clinician / S. Colagrande; S. Pradella ; S. Lucarini ; F. Marra. - In: DIGESTIVE AND LIVER DISEASE. - ISSN 1590-8658. - STAMPA. - 44:(2012), pp. 363-368. [10.1016/j.dld.2011.10.026]

Availability:

This version is available at: 2158/595424 since:

Published version:

DOI: 10.1016/j.dld.2011.10.026

Terms of use:

Open Access

La pubblicazione è resa disponibile sotto le norme e i termini della licenza di deposito, secondo quanto stabilito dalla Policy per l'accesso aperto dell'Università degli Studi di Firenze (https://www.sba.unifi.it/upload/policy-oa-2016-1.pdf)

Publisher copyright claim:

(Article begins on next page)

05 May 2023

Digestive and Liver Disease 44 (2012) 363–368

Contents lists available at SciVerse ScienceDirect

Digestive and Liver Disease

journal homepage: www.elsevier.com/locate/dld

Review article

Transient Hepatic Parenchymal Enhancement detected at dynamic imaging: A short instruction manual for the clinician☆

Stefano Colagrande[a,*], Silvia Pradella[b], Silvia Lucarini[b], Fabio Marra[c,d,**]

[a] Dipartimento di Fisiopatologia Clinica-Radiodiagnostica, University of Florence, Italy
[b] Dipartimento di Diagnostica per Immagini, Azienda Ospedaliero-Universitaria Careggi, Florence, Italy
[c] Dipartimento di Medicina Interna, University of Florence, Italy
[d] Centro di Ricerca, Trasferimento ed Alta Formazione DenoTHE, University of Florence, Italy

ARTICLE INFO

Article history:
Received 19 April 2011
Accepted 30 October 2011
Available online 8 December 2011

Keywords:
Biliary obstruction
Blood perfusion
Hepatocellular carcinoma
Hepatic metastases

ABSTRACT

Basic knowledge in the interpretation of hepatic imaging is essential for the clinical hepatologist. In recent years, the availability of dynamic imaging studies of the liver using computed tomography or magnetic resonance has led to appreciate the importance of early changes in arterial perfusion for the interpretation of hepatic lesions. Transient Hepatic Parenchymal Enhancement (THPE) is defined as a normal area of liver parenchyma that enhances after injection of contrast agent during the arterial phase of perfusion. Appearance of this sign is mostly associated with a reduction in portal perfusion or with inflammation, and appears in different morphologic patterns. THPE should not be considered a radiological artefact, and its interpretation is essential to avoid misclassification of hepatic lesions that may have clinical significance, such as hepatocellular carcinoma or hepatic metastases. In this short review we provide essential information on the causes, pathophysiology and morphology of THPE, and discuss the relevance of these findings in a clinical perspective.

© 2011 Editrice Gastroenterologica Italiana S.r.l. Published by Elsevier Ltd. All rights reserved.

1. Introduction

Detection of hepatic nodular lesions and their correct diagnosis is an everyday challenge for the clinical hepatologist. This is of particular relevance in the setting of a cirrhotic liver, where hepatocellular carcinoma (HCC) represents a leading complication and an increasingly observed cause of death [1]. According to the existing guidelines, imaging plays a critical role in the definition of a nodular lesion as HCC in the context of a cirrhotic liver [2]. These considerations imply that the clinical hepatologist acquires at least some knowledge of the criteria that are necessary for the interpretation of liver computed tomography (CT) or magnetic resonance (MR) dynamic imaging. In fact, correct understanding of imaging findings often needs close cooperation between radiologists and clinicians, especially in the setting of cirrhosis, where suspicion of malignancy is common.

Due to its high temporal resolution (speed), multi-detector CT allows to acquire images of large portions of the body in a single breath-hold. In a typical dynamic study of the abdomen, and particularly of the liver, the unenhanced scan is usually followed by two (or three) additional scans acquired during the arterial and portal venous phases (approximately 30 and 70 s following administration of contrast medium, respectively), to obtain information about arterial and portal perfusion of the hepatic parenchyma. Dynamic imaging techniques have identified a new phenomenon related to changes in arterial perfusion of the liver, which occurs rather frequently in CT imaging (between 9.3% and 15% of all exams performed on the abdomen) [3]. This phenomenon was first described by Itai et al. [4] who named it "Transient Hepatic Attenuation Difference" (THAD); it represents a very common cause of diagnostic uncertainty, often leading to additional imaging studies and possibly even to liver biopsy. In this paper we review the biological and clinical significance of these hepatic arterial phenomena, focusing on the possible diagnostic pitfalls.

2. Definition

Since the first description by Itai in CT scans, a THAD has been defined as 'a normal area of liver parenchyma that enhances after injection of contrast agent during the arterial phase of

☆ *Grant support*: Work in Dr. Marra's laboratory is supported by grants from the Istituto Toscano Tumori (ITT) and Associazione Italiana per la Ricerca sul Cancro (AIRC).

* Corresponding author at: Università di Firenze, Dipartimento di Fisiopatologia Clinica - Unità di Radiodiagnostica, Largo Brambilla 3, 50134 Florence, Italy. Tel.: +39 055 4377673; fax: +39 055 431970.
** Corresponding author at: Dipartimento di Medicina Interna, Largo Brambilla 3, I-50134 Florence, Italy. Tel.: +39 055 4271087; fax: +39 055 417123.
E-mail addresses: stefano.colagrande@unifi.it (S. Colagrande), f.marra@dmi.unifi.it (F. Marra).

1590-8658/$36.00 © 2011 Editrice Gastroenterologica Italiana S.r.l. Published by Elsevier Ltd. All rights reserved.
doi:10.1016/j.dld.2011.10.026

perfusion'. The same phenomenon is defined as Transient Hepatic Intensity Difference (THID) in MR imaging [5,6]. The term THPE (Transient Hepatic Parenchymal Enhancement) has therefore been created to encompass both THAD and THID [7]. TPHE indicates an increase in arterial perfusion, due to a variation of the normal hepatic blood supply. THPE is generally observed as an area of hyperdensity/hyperintensity (then with appearance brighter than surrounding areas) in the parenchyma in the arterial phase, which tends to disappear during the portal phase [8,9].

To understand the basis of THPE, it is important to consider the peculiarity of the hepatic vascular system. The liver receives a dual blood supply from the portal vein (about 75%) and the hepatic arteries (about 25%). These two systems are interconnected through the trans-sinusoidal, the transvasal and the peribiliary plexus, which provide a compensatory system to ensure a constant flow of blood through the hepatic parenchyma, regardless of variations that may occur in either the arterial or venous supplies [10,11]. The autonomic nervous system and soluble factors regulate flow through these vessels. It should be underscored that THPE can be seen as the imaging equivalent of a phenomenon known as "arterial buffer response", which is activated by the liver based on the requirement of oxygen and metabolites. As a consequence of a decrease in portal supply, an increase in arterial flow is generated and becomes detectable in the arterial phase of CT imaging [11,12].

3. Pathophysiology and imaging findings

From a pathogenetic point of view, THPE can be divided into two major categories: (1) those based on arterial hyperperfusion secondary to a reduction of portal perfusion and (2) those in which the arterial hyperperfusion is primary, independently of the occurrence of a portal hypoperfusion.

Portal hypoperfusion may be caused by obstruction, thrombosis, compression or infiltration of a branch of the portal vein, as occurs in case of compression *ab extrinseco*, e.g. by a nodular lesion or a haematoma. Hypoperfusion may also be caused by flow diversion as occurs in arterio-portal shunts (APS), caused by small arterio-portal fistulas or by the presence of an abnormal afferent blood as in the case of venous or arterial anatomical variations. In these latter conditions arterial blood, with higher pressure, flows into the portal bed, with lower pressure, and blocks portal flow, thus triggering the arterial reaction and then generating the THPE.

This type of diversion can also take place in the territories of the "third hepatic inflow" which consists of the capsular veins, the Sappey paraumbilical veins, hilar and epiploic veins, suspensory ligament and diaphragmatic veins and the accessory cystic vein. These anomalous accessory veins enter the liver separately from the portal venous system and may act, accordingly to the pressure gradient, as anomalous supply or drainage vessels, providing some areas of the parenchyma, mainly located in segments I–IV. As a consequence, these venous systems work as a shunt between the systemic venous circulation and the sinusoids. In physiological conditions they represent a hepatopetal system, contributing no more than 2–3% of the hepatic blood flow. In the presence of portal hypertension, the intraportal pressure becomes higher than that of the systemic veins, and the "third" hepatic system becomes hepatofugal, allowing for the development of shunting systems in a cirrhotic liver [13–15].

A second type of THPE is independent of portal hypoperfusion and is mainly related to inflammation, as in the case of cholecystitis, abscesses or cholangitis. Inflammatory mediators may cause vasodilatation and increased vascular permeability in the nearby normal parenchyma. Once the inflammation is resolved, the THPE phenomena also disappear. Another type of primary THPE may be related to the presence of hypervascular benign tumours ('sump effect', see below).

Identification of THPE and increased knowledge of their different types is a major challenge for the clinician who has to interpret these findings. In the clinical setting, it is particularly important to evaluate the possibility that some of these pictures may be the expression of proliferative lesions or that herald their eventual appearance. A pivotal aspect is that a radiologist must describe the presence of a THPE, even though he/she is unable to determine its cause. On the other hand, the clinician should never consider THPE as an artefact, and these findings need to be placed in the framework of the clinical history and current conditions of the patient. In any case, a close interaction between the radiologist and the clinician is essential, in order to reduce the number of missed or wrong diagnoses and to warrant an appropriate follow-up to the patient.

Ideally, THPE should be classified according to their etiopathogenesis. However, this approach is impractical because morphology is the most appreciable characteristic of these arterial phenomena. Thus, this review is organized according to the imaging appearance of THPEs, distinguishing localized and diffuse forms of THPE (Table 1 and Fig. 1).

3.1. Localized THPEs

These are generally associated with portal hypoperfusion, and particularly with portal vein thrombosis. They appear mostly as triangular areas, and may also be associated with malignant or benign lesions, such as haemangioma or abscess [16,17]. Depending on the position of the lesion, the arterial area may be differently sized. Especially in case of triangular-shaped THPE, the radiologist should

Table 1
Classification of the different types of Transient Hepatic Parenchymal Enhancement.

Extension	Pathogenesis	Appearance	Predominant causes
Localized	Portal hypoperfusion	Triangular	• Benign or malignant focal lesion (rarely hidden nodule) • Arterio-portal shunt • Portal thrombosis
	Inflammation	Polymorphous	• Cholecystitis, pleuritis • Other causes: parenchymal injury, extrinsic compression, percutaneous biopsy or treatments. In these latter cases they present in various shapes, including triangular
	Primary hyperperfusion not linked to portal flow blockade ('sump effect')	Lobar or multi-segmental	• Large and benign focal lesions • Vascular variants (which can also induce polymorphous Transient Hepatic Parenchymal Enhancement)
Diffuse	Post-sinusoidal flow blockade	Patchy pattern	• Right heart failure • Budd–Chiari syndrome
	Pre-sinusoidal flow blockade	Central–peripheral pattern	• Thrombosis of portal trunk • Cirrhosis
	Peribiliary plexus impairment, frequently associated with inflammation	Peribiliary pattern	• Biliary tree dilation with or without inflammation (e.g. choledocholithiasis or cholangiocarcinoma, cancer of the head of pancreas)

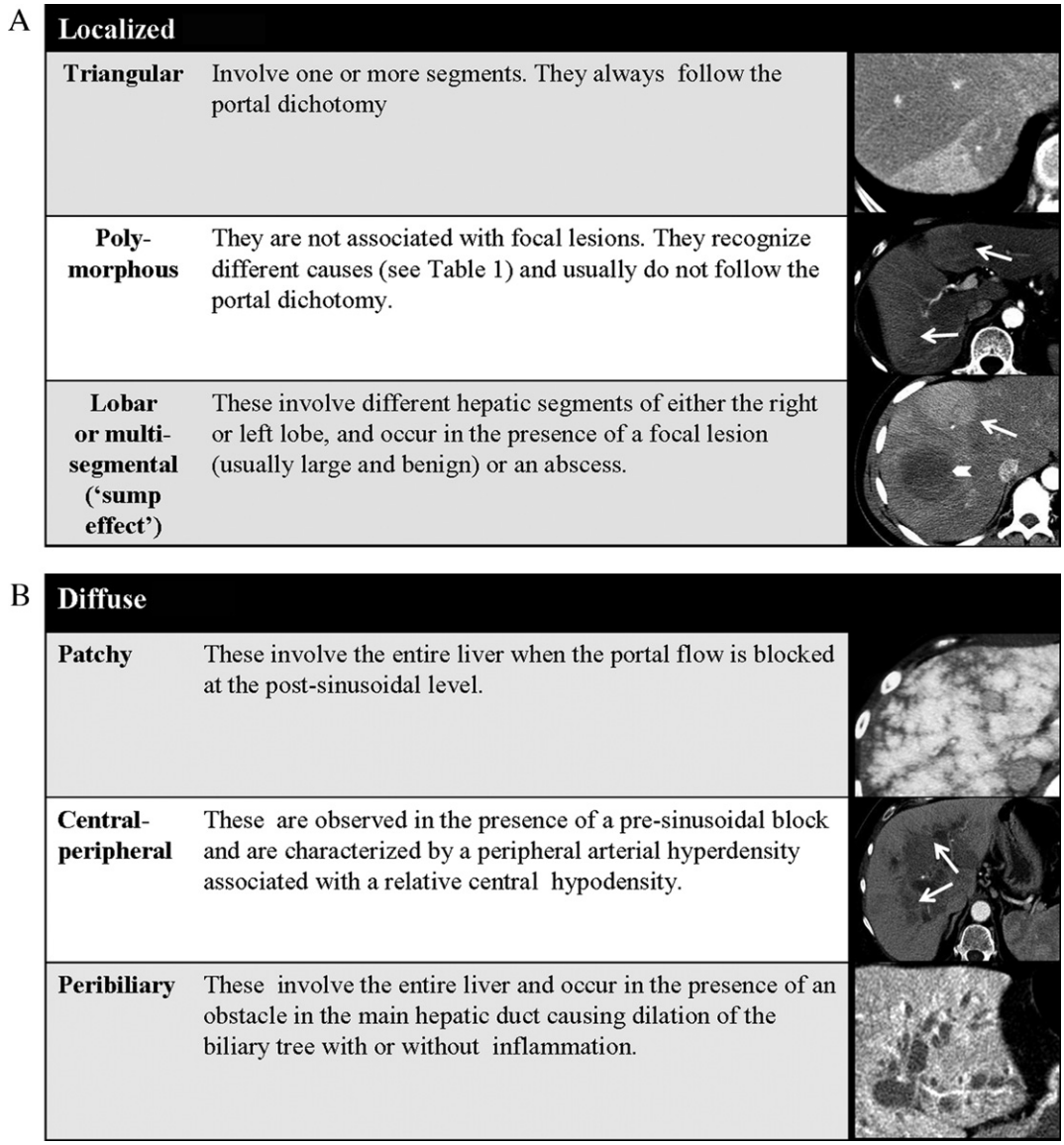

Fig. 1. Appearance of Transient Hepatic Parenchymal Enhancement. Top panel: examples of different types of localized Transient Hepatic Parenchymal Enhancement (THPE). Upper image: triangular THPE caused by a nodular lesion (not visible in the image provided); middle image: polymorphous THPE (arrows) associated with extrinsic compression of the right hepatic lobe caused by fluid collection; lower image: lobar THPE (arrow) caused by a hepatic abscess (arrowhead). Bottom panel: examples of different types of diffuse THPE. Upper image: patchy THPE in a patient with Budd–Chiari syndrome; middle image: central–peripheral phenomenon associated with portal vein thrombosis (not visible in the image provided); lower image: peribiliary pattern in a patient with biliary tree obstruction due to choledocholitiasis.

carefully look for the presence of a nodular lesion, which is usually placed within the THPE or at its apex. In fact, in some cases, the focal lesion may be very small or isodense to the area of THPE, and therefore difficult to identify. In this occurrence, a "hidden nodule" should be suspected, causing a portal compression, but not detectable because of being too small or devoid of a sufficient contrast difference with respect to the surrounding structures [18,19]. In all these cases a follow-up is strongly recommended, as it is possible that the focal lesion will appear with time (Fig. 2). A common condition that needs particular attention is the appearance of a triangular THPE in the context of a cirrhotic liver, especially when in proximity of the hepatic capsule where it can mimic a nodule due to the incidence of the X-ray beam (Fig. 3). In contrast to the typical CT appearance of HCC in cirrhosis (hyper in the arterial phase with portal washout), THPE do not show any washout during the portal phase. Also in this case, if a THPE is detected by CT scan, additional imaging studies such as MRI with liver-specific gadolinium chelates are necessary. In fact, typical HCC usually do not enhance in the hepatobiliary phase, whilst THPE shows a delayed enhancement equal to that of the surrounding parenchyma. This behaviour of THPE could mimic a well-differentiated HCC and therefore a close follow-up is mandatory. Thus, when a nodule without the typical HCC pattern is detected in a cirrhotic liver, the possibility of a pseudonodular THPE should be kept in mind [20]. THPE with pseudonodular appearance can be mainly found in subcapsular areas and in the district of the "third hepatic supply". It should be underscored that in these areas, arterial phenomena can be observed also in non-cirrhotic livers, especially after chemotherapy, which can damage minor portal vessels and facilitate blood inflow through systemic veins (i.e. the "third inflow") (Fig. 4). Clearly, appearance of these pseudonodular lesions in a patient undergoing chemotherapy may be challenging as they may raise the suspicion of a liver metastasis.

Sometimes localized THPEs are not triangular but polymorphous and/or round-shaped. These latter are usually not associated with focal lesions and are mainly caused by APS, parenchymal

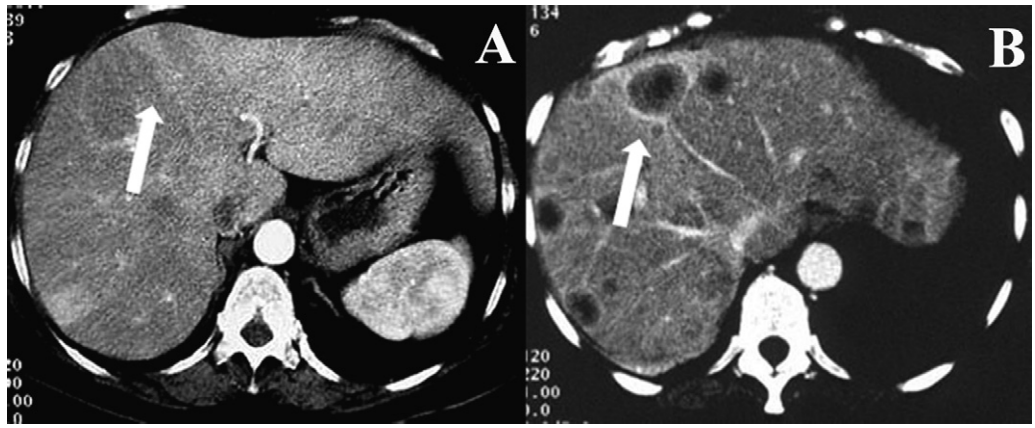

Fig. 2. Example of a 'hidden nodule'. (A) Presence of multiple Transient Hepatic Parenchymal Enhancements (THPE) (arrow for an example) in a patient without evidence of nodular lesions. (B) In a follow-up computed tomography scan after 6 months, there is clear evidence of multiple metastases in areas where THPE were previously detected (arrow).

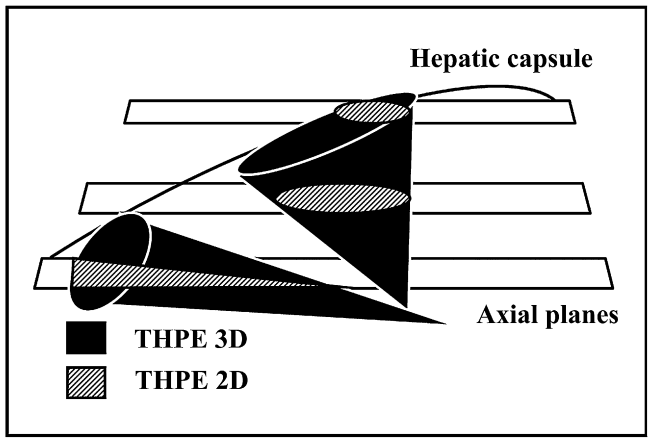

Fig. 3. Diagram of the nodular appearance of a Transient Hepatic Parenchymal Enhancement. The drawing shows how in axial images the shape of a Transient Hepatic Parenchymal Enhancement (THPE) depends on the plane orientation with respect to the arterialized area. This may mimic a nodular lesion.

Fig. 5. Example of a localized, polymorphous Transient Hepatic Parenchymal Enhancement. Arterial phase-computed tomography image showing an arterial phenomenon with a polymorphous pattern (arrow) in a patient with cholecystitis (inflammatory pathogenesis).

injury, extrinsic compression or inflammation (Fig. 1) [21]. A particular condition where multiple APS may be present is hereditary haemorrhagic telangiectasia (Rendu–Osler–Weber disease) [7]. Anomalous arteries or accessory veins, as in the "third hepatic supply" (see above) are another cause of altered blood supply that may lead to appearance of this type of THPE.

There are polymorphous THPEs which are not closely related to portal hypoperfusion. These forms of THPE are prevalently linked to inflammation of bile ducts and/or adjacent organs (cholecystitis, pancreatis, abscesses) [6] (Fig. 5). These include conditions defined in other studies as "inflammatory hepatic artery hyperaemia" and increased blood flow from a dilated aberrant cystic vein [22]. In these cases, arterial phenomena are located around the inflamed area. Their morphogenesis is related to the spread of inflammatory mediators to the parenchyma, by contiguity, although increased

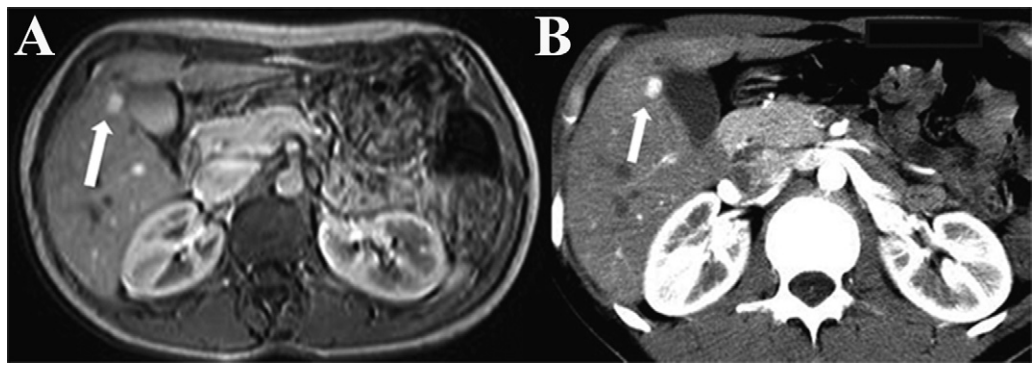

Fig. 4. Transient Hepatic Parenchymal Enhancement in the district of the 'third hepatic supply'. Arterial phases of magnetic resonance imaging (A) and computed tomography scan (B) show the presence of a nodular Transient Hepatic Parenchymal Enhancement near to the gallbladder (arrows) as a typical area of 'third inflow'.

arterial flow may also be secondary to portal inflow reduction due to interstitial oedema [10].

A third, less common type of localized THPE, not related to portal hypoperfusion, is the so-called "sump effect", also known as "syphoning phenomenon", which is associated with haemangiomas, focal nodular hyperplasia, or hypervascular tumours, usually larger than 3 cm and benign [23]. These tumours can markedly increase the arterial blood supply to the lobe or segment where it is contained, resulting in a transiently higher arterial attenuation in the lobar area surrounding the tumour. In contrast, the contralateral lobe receives a less abundant arterial blood supply and shows lower arterial attenuation. Another related phenomenon, although rarely occurring, is the 'steal phenomenon', the explanation of which exceeds the scopes of this review [7].

3.2. Diffuse THPEs

These types of THPE involve the entirety or the most part of the liver with a patchy, central–peripheral or peribiliary pattern (Table 1). They are usually associated with portal and/or biliary obstruction, and the imaging appearance depends on both the level and the type of obstruction [5,6,18,24]. A patchy pattern is usually due to post-sinusoidal obstruction of the portal flow, which can occur at the level of the hepatic veins, such as in case of heart failure, Budd–Chiari syndrome or inferior vena cava obstruction syndrome. In these conditions, occlusion of the hepatic veins results in increased sinusoidal pressure and reverses the pressure gradient between the sinusoidal and portal veins. The portal vein then becomes a draining vein, causing an increase in arterial blood flow, and resulting in a functional APS [25,26]. At dynamic imaging, this appears as an enhancement of the central part of the hepatic parenchyma (centrilobular enhancement) with a mottled/marbled appearance. This pattern is evident in the arterial phase, but is typically maintained in the portal phase (Fig. 1).

In sinusoidal and pre-sinusoidal obstruction, the interconnecting shunts between the arterial and the portal systems play an important role, in particular the opening of the peribiliary plexus, which determines the central–peripheral pattern, i.e. an arterial enhancement of the peripheral subcapsular parenchyma with relative hypodensity of the perihilar area. The block occurs either at the portal trunk (before the sinusoids), as in portal vein thrombosis, or at sinusoidal level, as in cirrhosis [7]. Portal flow usually remains adequate in the central area of the liver (segments I and IV), and in segments II and III, whilst in the periphery its relative deficiency leads to an increase in arterial flow, with opening of shunts at the level of the peribiliary plexus.

A pattern intermediate between the patchy and the central–peripheral phenomenon, with variable expression, may be noted during veno-occlusive disease, more recently named sinusoidal obstruction syndrome. This describes a non-thrombotic obstruction of the hepatic sinusoids with or without venular involvement, based on endothelial toxicity mainly due to chemotherapy and/or radiotherapy. The consequent congestion of the liver parenchyma may lead to a light patchy pattern with linear hypodensity due to sinusoidal wall oedema. Nonetheless, the diagnosis of this condition is based on liver biopsy [27].

A peribiliary pattern may be observed in the case of long-standing bile duct dilation (e.g. due to choledocholithiasis, pancreatic cancer or ampulloma). Increased pressure causes compression of the peribiliary plexus, which surrounds the bile duct and lacks a muscular wall. This may cause a reduction of blood flow from the portal vein to the sinusoids, resulting in an arterial compensation and a diffuse and irregular enhancement of the peribiliary parenchyma (Fig. 6). If the cause of obstruction is not removed, persistence of cholestasis leads to parenchymal atrophy, especially in the presence of portal vein obstruction [28] whilst if the block is removed, the THPE phenomena disappear [18,29]. This pattern may be present also in the case of cholangitis, when the peribiliary plexus is blocked because of the involvement by the inflammation. This can be of clinical utility, supporting the diagnosis of cholangitis, that may be difficult in the absence of bile duct dilation.

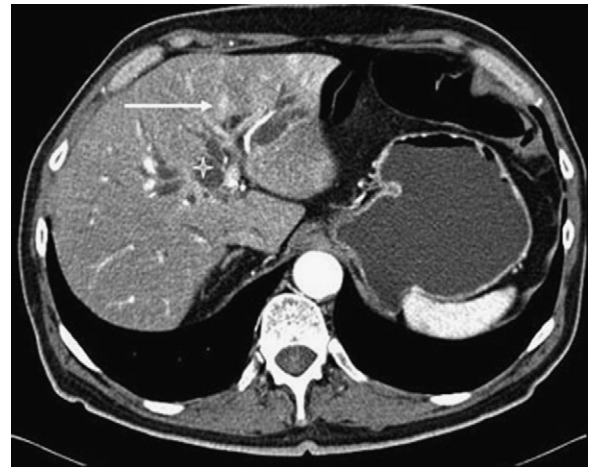

Fig. 6. Example of a diffuse, peribiliary. Transient Hepatic Parenchymal Enhancement. Arterial phase-computed tomography image showing a Transient Hepatic Parenchymal Enhancement with peribiliary pattern (arrow) in a patient with dilatation of the intrahepatic branches of the biliary tree (star) associated with stenosis of the distal tract of the common bile duct.

This type of THPE usually presents in a diffuse form, but it may be appearing also in a triangular shape if only a branch of the biliary tree is involved. In these latter cases, if CT scan is not sufficient to detect the cause of obstruction, additional investigation with ultrasound and/or MRI should be performed. After removal of the obstruction (such as in case of biliary stones), follow-up is mandatory because THPE tend to disappear in these cases. In contrast, in the case of malignant disease, THPE tend to persist and additional efforts must be put to identify its cause.

4. Evolution of THPE

In general, if the cause of THPE is removed, arterial phenomena rapidly disappear. However, even if the cause persists, imaging alterations tend to become less evident with time and eventually disappear within months. This may be explained by the fact that in normal conditions the liver needs low oxygen tension and high levels of nutrients. When these conditions are not met, as in the case of a persistently increased arterial blood flow, the liver parenchyma undergoes metabolic changes that present as an area of hypodensity on imaging. This is likely due to the presence of oedema, fibrosis and/or depletion of hepatocytes in the underlying parenchyma [7,25,30].

5. Conclusion

Several studies in recent years have highlighted the potential relevance of THPE phenomena observed by hepatic imaging performed by CT scan or MR imaging. The clinical relevance of these forms is based on the possibility to be misdiagnosed as cancerous lesions, to hide an underlying cancer, or to be expression of severe hepatic diseases. In all these cases, incorrect recognition and classification of a THPE will prompt costly and possibly unnecessary tests and will be a source of considerable distress for the patient. Thus, a close collaboration between the radiologist and the clinician is necessary to discuss the clinical and imaging findings of each controversial case, thus increasing the possibility to reach a correct diagnosis.

Conflict of interest statement

The authors have no conflicts of interest to disclose.

List of abbreviations

APS, arterio-portal shunt; CT, computed tomography; HCC, hepatocellular carcinoma; MR, magnetic resonance; THAD, Transient Hepatic Attenuation Difference; THID, Transient Hepatic Intensity Difference; THPE, Transient Hepatic Parenchymal Enhancement.

References

[1] Yang JD, Roberts LR. Hepatocellular carcinoma: a global view. Nat Rev Gastroenterol Hepatol 2010;7:448–58.
[2] Bruix J, Sherman M, American Association for the Study of Liver Diseases. Management of hepatocellular carcinoma: an update. Hepatology 2011;53:1020–2.
[3] Golfieri R, Centi N, Fusco F, et al. Pseudolesioni da flusso e vere lesioni nodulari flusso-dipendenti. In: Strambini T, editor. Diagnostica per immagini delle malattie del fegato e delle vie biliari. Milano: Masson; 2005. p. 373–402.
[4] Itai Y, Moss AA, Goldberg HI. Transient hepatic attenuation differences of lobar or segmental distribution detected by dynamic computed tomography. Radiology 1982;144:835–9.
[5] Colagrande S, Centi N, Galdiero R, et al. Transient hepatic intensity differences. Part I. Those associated with focal lesions. AJR Am J Roentgenol 2007;188:154–9.
[6] Colagrande S, Centi N, Galdiero R, et al. Transient hepatic intensity differences. Part II. Those not associated with focal lesions. AJR Am J Roentgenol 2006;188:160–6.
[7] Quiroga S, Sebastià C, Pallisa E, et al. Improved diagnosis of hepatic perfusion disorders: value of hepatic arterial phase imaging during helical CT. Radiographics 2001;21:65–81.
[8] Chen WP, Chen JH, Hwang JI, et al. Spectrum of transient hepatic attenuation differences in biphasic helical CT. AJR Am J Roentgenol 1999;172:419–24.
[9] Gryspeerdt S, Van Hoe L, Marchal G, et al. Evaluation of hepatic perfusion disorders with double-phase spiral CT. Radiographics 1997;17:337–48.
[10] Colagrande S, Carmignani L, Roselli G, et al. Arterial hepatic sectorial enhancement [ASE] as a sign of acute portal thrombosis three case reports. Radiol Med 1998;96:639–41.
[11] Kawasaki T, Carmichael FJ, Saldivia V, et al. Relationship between portal venous and hepatic arterial blood flows. Spectrum of response. Am J Physiol 1990;259:G1010–8.
[12] Gulberg V, Haag K, Roessle M, et al. Hepatic arterial buffer response in patients with advanced cirrhosis. Hepatology 2002;35:630–4.
[13] Hashimoto M, Heianna J, Tate E, et al. Small veins entering the liver. Eur Radiol 2002;12:2000–5.
[14] Breen D, Rutherford E, Stedmann B, et al. Intrahepatic arterioportal shunting and anomalous venous drainage: understanding the CT features in the liver. Eur Radiol 2004;14:2249–60.
[15] Gallego C, Velasco M, Marcuello P, et al. Congenital and acquired anomalies of the portal venous system. Radiographics 2002;22:141–59.
[16] Giovagnoni A, Terilli F, Ercolani P, et al. MR imaging of hepatic masses: diagnostic significance of wedge-shaped areas of increased signal intensity surrounding the lesion. AJR Am J Roentgenol 1994 Nov;163:1093–7.
[17] Yamasaki M, Furukawa A, Murata K, et al. Transient hepatic attenuation difference (THAD) in patients without neoplasm: frequency, shape, distribution, and causes. Radiat Med 1999;17:91–6.
[18] Colagrande S, Carmagnini L, Pagliari A, et al. Transient hepatic attenuation differences (THAD) not connected to focal lesions. Radiol Med 2002;104:25–43.
[19] Brink JA, Increased CT. Contrast enhancement of "normal" hepatic parenchyma may herald occult "metastases". Radiology 1997;205:37–8.
[20] Colagrande S, La Villa G, Bartolucci M, et al. Spiral computed tomography versus ultrasound in the follow-up of cirrhotic patients previously treated for hepatocellular carcinoma: a prospective study. J Hepatol 2003;39:93–8.
[21] Ahn JH, Yu JS, Hwang SH, et al. Nontumorous arterioportal shunts in the liver: CT and MRI findings considering mechanisms and fate. Eur Radiol 2010;20:385–94.
[22] Ito K, Awaya H, Mitchell DG, et al. Gallbladder disease: appearance of associated transient increased attenuation in the liver at biphasic, contrast-enhanced dynamic CT. Radiology 1997;204:723–8.
[23] Colagrande S, Centi M, Pradella S, et al. Transient hepatic attenuation differences and focal liver lesions: sump effect due to primary arterial hyperperfusion. JCAT 2009;33:259–65.
[24] Kobayashi S, Ng CS, Kazama T, et al. Hemodynamic and morphologic changes after portal vein embolization: differential effects in central and peripheral zones in the liver on multiphasic computed tomography. J Comput Assist Tomogr 2004;28:804–10.
[25] Itai Y, Matsui O. Blood flow and liver imaging. Radiology 1997;202:306–14.
[26] Kim TK, Choi BI, Han JK, et al. Nontumorous arterioportal shunt mimicking hypervascular tumor in cirrhotic liver: two-phase spiral CT findings. Radiology 1998;208:597–603.
[27] Mahgerefteh SY, Sosna J, Bogot N, et al. Radiologic imaging and intervention for gastrointestinal and hepatic complications of hematopoietic stem cell transplantation. Radiology 2011;258:660–71.
[28] Hann LE, Getrajdman GI, Brown KT, et al. Hepatic lobar atrophy: association with ipsilateral portal vein obstruction. AJR Am J Roentgenol 1996;167:1017–21.
[29] Colagrande S, Centi N, La Villa G, Villari N. Transient hepatic attenuation differences. AJR Am J Roentgenol 2004;183:459–64.
[30] Golfieri R, Gavelli G. Liver hemodynamic changes, pseudolesions and benign nodular flow-dependent lesions their pathogenesis and spiral computed tomographic radiological aspects. Radiol Med 2000;99:233–49.